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Foreign Animal Disease Report

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Current Events

Emergency Disease Investigations, 1986

During fiscal year 1986 (Oct. 1, 1985, through Sept. 30, 1986), 165 foreign animal disease (FAD) diagnostic investigations were conducted in the United States and Puerto Rico, including 115 investigations of suspected vesicular conditions.

The first isolation of New Jersey type vesicular stomatitis (NJVS) virus this year was made at the Veterinary Services (VS) Foreign Animal Disease Diagnostic Laboratory (FADDL) in Plum Island, N.Y., from a Montezuma County, Colorado, bovine tissue submission collected July 18, 1986. The following table summarizes the results of laboratory tests of suspected vesicular diseases during fiscal year 1986:

State	Total Number of Specimens Tested	
	NJVS Virus Isolated	NJVS Antibodies Identified
Arizona	0	1
Colorado	6	27
New Mexico	0	15
Utah	2	0

Fifteen investigations of suspected hog cholera (HC) or suspected African swine fever (ASF) were completed in response to reports of either suspicious clinical signs or suspicious reactions to laboratory tests of serums collected during routine surveillance of slaughtering establishments. All investigations were HC/ASF negative. About 20,000 swine blood samples from pigs slaughtered in

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Massachusetts, New Hampshire, New Jersey, Texas, and Puerto Rico were tested at the National Veterinary Services Laboratories (NVSL) in Ames, Iowa. This testing regularly supplements our primary defenses against possible incursions of African swine fever or hog cholera.

This past summer, public health researchers working on Acquired Immune Deficiency Syndrome in Belle Glade, Fla., excited the news media when they sampled sick swine in the area and reported evidence of African swine fever (ASF). As soon as the Animal and Plant Health Inspection Service (APHIS) became aware of these reports, the pigs were quarantined under State authority. All adult hogs on three farms were examined and samples were sent to FADDL. Exhaustive testing of 126 serum samples, 43 tonsil biopsies, and other tissue specimens proved that neither ASF virus nor antibody was present. Specimens carried to Pirbright, England, by the researchers were also tested and found to be free of ASF virus.

Four VS veterinarians were sent to Mexico to gain firsthand experience in the diagnosis of hog cholera as part of ongoing efforts to maintain a cadre of experienced hog cholera diagnosticians.

On Nov. 13, 1985, velogenic viscerotropic Newcastle disease (VVND) virus was isolated from a yellow nape Amazon parrot in the State of Washington. The virus was also isolated from pet birds on the premises of the dealer from which the parrot was purchased in California. All contaminated birds were depopulated.

On March 31, 1986, VVND virus was isolated from a yellow nape parrot located at Belvidere, N.J. The infected bird had been purchased March 15, 1986, from a Cinnaminson, N.J., pet bird dealer. The dealer's 2,414 birds were depopulated on April 2, 1986, to prevent further spread of the virus. Over 300 shipments of pet birds from the Cinnaminson dealer were traced to 12 States. An additional infected dealer's premises was identified at Toms River, N.J. Three additional cases of VVND in yellow napes were reported in California during March and April. The origin of the infection in these outbreaks was not found; however, circumstantial evidence suggest that smuggled birds from Mexico were responsible.

Two VVND virus-infected, smuggled cockatoos were intercepted at the St. Thomas, U.S. Virgin Islands, airport. Both were seized and both died of VVND soon thereafter.

No VVND was identified in commercial poultry this year and the disease was again successfully excluded from pet bird trade channels.

During fiscal year 1986, a national survey for

Nematodirus battus in sheep flocks was conducted. Thirty three thousand and six hundred one (33,601) fecal specimens from 49 States, the U.S. Virgin Islands, and Puerto Rico were examined at NVSL. Sheep were found to be affected on 33 premises in Oregon, Washington, Maryland, Vermont, and New York. (Dr. A. E. Hall, 301-436-8073)

READI System Revised

The VS Recorded Emergency Animal Disease Information (READI) computer system has been extensively revised for user-friendly data entry and retrieval at any telephone-accessible location. The changes will permit more rapid and convenient management of data during routine investigations of suspected foreign animal diseases, as well as during a foreign animal disease task force operation. (Dr. E. I. Pilchard, 301-436-5959)

Vesicular Stomatitis Field Studies, 1982-1985

Many aspects of the biology and epidemiology of vesicular stomatitis (VS) remain a mystery despite years of study and speculation (see review in 10-3). VS is caused by two antigenically and epidemiologically distinct viruses: VS Indiana (VSI) and VS New Jersey (VSNJ). Both appear annually in tropical and subtropical areas of the Americas, whereas in the midwestern and western United States infrequent epizootic waves appear at intervals of up to 10 years. VSNJ has been the predominant serotype in epizootic vesicular disease in the United States for the last 20 years.

The disease appears to spread up and down valleys and river drainages in wet pastures, suggesting insect transmission. The main reasons for suspecting arthropod transmission are: (1) outbreaks occur in the summer at the height of the insect season, (2) VS viruses replicate in and can be transmitted by arthropods, and (3) VS virus has occasionally been isolated from field-collected arthropods. Despite these observations, doubt remains regarding the role of arthropods in natural transmission cycles. Virus can seldom be isolated from the blood of an infected animal, and low-titered viremias insufficient to infect biting arthropods are the rule in experimental studies. During the 1982-83 epizootic, 38 VSNJ virus isolations were made from biting and nonbiting flies. The former included four isolations from Culicoides midges and two from Simulium blackflies. Isolation from nonbiting flies included 27 from muscoid flies (Musca domestica and M. autumnalis), 4 from Anthomyidae, and 1 from Hippelates eye gnats. These insects were collected on premises with active clinical cases. Certainly, arthropods could play a role in mechanical transmission during an epizootic because infected cows and horses may have large quantities of virus in saliva or on surfaces of affected mucous membranes to which arthropods are attracted. Simulium and Culicoides may feed on blood or secretions and the nonbiting flies on secretions around the mouths of livestock and then transfer contaminated saliva or vesicular fluid to susceptible animals. We

observed many flies transported in the cab of milk trucks going from farm to farm. Use of air conditioning and closed windows might reduce this risk.

It appears to be generally true that pastured animals are more frequently affected by the disease than animals in stables, barns, or dry field lots (Jonkers, A. H., Am. J. Epidemiol., 86: 288-291, 1967). A study conducted on a horse ranch in Loveland, Colo., during the 1982 outbreak showed disease in horses in a pasture that spread to animals in other pastures, and finally affected horses in barns and corrals. The incidence of inapparent infection was higher among animals in barns than among pastured animals. These findings suggest the occurrence of an environmental risk factor underlying disease expression, such as exposure to rough fodder causing mucosal abrasions. Thistles and sharp grasses could predispose to minute wounds and abrasions and allow for virus entry on contact with a contaminated animal, object, or arthropod. The progressive circumferential and centrifugal movement of virus on this farm was consistent with transmission by arthropods, although other means cannot be ruled out (Webb, P. A., et al., Am. J. Trop. Med. Hyg., in press, 1986). Certain arthropods, including Similium, Culicoides, and some mosquitoes, would be expected to feed much more readily on animals in pastures than in barns.

Contact transmission of virus between domestic animals was documented in 1982. Spread by cows dispersed from a dairy sale in Colorado and, subsequently, from heifers imported to California from Idaho. The view that outbreaks in the Western United States occur due to introduction of the virus from areas of enzootic activity, particularly the bordering Mexican States of Sonora and Chiapas, has been supported by RNA oligonucleotide fingerprinting of VSNJ strains from the United States and Mexico in 1982 and 1985 (Nichol, S. T., J. Virol., in press, 1986). Nichol found that VSNJ strains from Colorado, Idaho, Wyoming, California, New Mexico, and Nebraska in 1982-83 were identical to or very closely related to each other and to a 1982 strain from Veracruz, Mexico, but distant from the historical Ogden and Hazelhurst strains and from strains from the Southeastern United States (Ossabaw enzootic focus). Two 1985 strains from Sonora and Chihuahua, Mexico, were shown to be identical to six isolates from Arizona, New Mexico, and Colorado (from cattle and horses) but distant from the 1982-83 strains. This appears to point to known enzootic areas of VS activity in Mexico as the source of the 1982 and 1985 epizootic strains.

Serological evaluation of animals on infected premises requires tests to determine recent infection. High neutralizing (N) antibody titers persist for at least 4 to 5 years, particularly in equines, in titers up to

1:163,840 (Webb, P. A., et al., Am. J. Trop. Med. Hyg., in press, 1986). The complement fixation (CF) test or IgM antibody capture enzyme-linked immunosorbent assay are both methods in current use to determine if infection has occurred within the past 2 months. Although N antibody can also be detected in milk samples, titers are considerably lower than in serum samples; however, milk samples are easily obtained without interrupted milking schedules. The milk testing method may provide valuable seroepidemiological evidence of past exposure to VS viruses.

Naturally occurring antibody in wildlife has been observed frequently. Antibodies were found in elk bled at high elevations in the Rocky Mountain National Park, Colorado, during 2 years preceding the 1982 epizootic. Seroprevalence rates increased between 1980 and 1981, suggesting "amplification" of virus prior to the 1982 epizootic season (Webb, P. A., et al., J. Wildl. Dis., in press, 1986). This finding suggests the virus has been repeatedly introduced or that there is a local virus reservoir. Pronghorn antelope and mule deer have high seroprevalence rates and frequently graze with domestic animals.

Studies on vaccinated herds have been limited, and the possibility that vaccine-induced N antibody is protective needs further study. Using a formalin-killed VSNJ vaccine, animals within a Colorado State University dairy herd attained a very low titered N antibody response, with peak titers (160-640) occurring about 7 weeks after inoculation and falling sharply (to 40) by 12 weeks (Gearhardt, M. A., et al., J. Am. Vet. Med. Assoc., in press, 1986). In a comparison of three dairy herds in the Pueblo, Colo., area in 1985, overt oral lesions occurred in 95 percent of one herd and peak N antibody titers reached 40,960, whereas in a dairy with only 25 percent of the herd infected (with mostly teat lesions), titers were 640. The third dairy had vaccinated their herd during the 3 preceding years; overt disease was minimal, and peak antibody titers were similar (640).

In future outbreaks, whether of epizootic proportions or limited to a few premises, field investigations need to focus on certain issues. Is the disease really irregularly distributed, skipping some herds while affecting others? It may be that overt disease expression is limited, whereas serological evidence is widespread. This must be substantiated by tests for recent infection.

If the pasture is a main disease-producing unit, intensive ecological studies may help us decide why.

Epidemiological enquiries regarding movement of animals, introduction of new animals, cograzing with wildlife, and

recent stress factors need to be examined each time disease appears. It is particularly important to ascertain the exact number of animals on each premise, their vaccine status, and serological status.

Many questions need careful notation. Some interesting suggestions made by Dr. A. H. Jonkers are these: In herds where teat lesions predominate, is this related only to milking techniques or to cows lying in the pasture? Are the teat lesions in juxtaposition (left front, right rear), which would be expected if the animal picks up virus by lying down on it? Do cows lie predominantly on one side? Cows and horses eat differently; horses gather grasses with the lips and bite it off, and cattle wrap the tongue around the grass and tear it off. Does the site of the lesions reflect this? On farms that experience reinfection, are the same animals reinfected? (Dr. T. P. Monath and Dr. P. A. Webb, U.S. Department of Health and Human Services, Division of Vector-borne Viral Diseases, P.O. Box 2087, Ft. Collins, Colorado 80522-2087, 303-221-6404)

Zoological Animal Regulation Proposed

During recent years, increased trade in zoological animals, and hence the increase in interstate movement, has magnified the potential for disease dissemination. Past experiences with tuberculosis in bison and elk, exotic Newcastle disease in pet birds, lethal avian influenza in fancy poultry and game birds, and foreign ticks on imported rhinoceroses have caused livestock and poultry health officials considerable anxiety. Since many zoological animals are exotic to the United States, their commercial trade has substantial foreign animal disease implications.

Last year the United States Animal Health Association (USAHA) formally requested that the Animal and Plant Health Inspection Service (APHIS), United States Department of Agriculture (USDA), consider the development of a model State regulation for control of zoological animals. Through a cooperative agreement, APHIS enlisted the help of the Southeastern Cooperative Wildlife Disease Study (SCWDS), College of Veterinary Medicine, University of Georgia, to evaluate the current situation and draft a model State regulation.

Zoological animals can be broadly defined as recently captured native and non-native (exotic) wild animals, native and non-native wild animals raised in captivity, and wild forms of domesticated species. Conventional domesticated animals and some laboratory-adapted strains of wild animals are not included in this definition.

Existing regulations concerning the ownership, sale, and transportation of zoological animals are generally inadequate and are fragmented among 50 State departments of agriculture, 50 State departments of natural

resources, the USDA, and the U.S. Department of the Interior.

A draft copy of a model State regulation was prepared that addresses ownership, sale, transportation, release, disease prevention, public safety, environmental protection, and animal welfare. Copies were submitted to Veterinary Services, APHIS, USDA, for review. At that time, APHIS officials requested that the model regulation also be made available for review by interested committees of the USAHA. These committees were asked to submit copies of the draft regulation to other organizations, particularly the International Association of Fish and Wildlife Agencies (IAFWA) and the National Association of State Public Health Veterinarians (NASPHV). Other interested organizations and individuals may request a copy of the model regulation by writing to SCWDS, University of Georgia, Athens, GA 30602. Comments and suggestions will be considered in the preparation of a revised draft, to be completed by early 1987. It is anticipated that the revised draft model State regulation will be offered to the USAHA, IAFWA, and NASPHV for endorsement. An endorsed model State regulation would then be distributed to State departments of natural resources, agriculture, and public health for their use. (Dr. V. F. Nettles and Mr. P. K. Swiderek, 404-542-1741).

Foreign Animal Disease Update

From November 26, 1984, through August 1985, 142 outbreaks of foot-and-mouth disease (FMD) serotype A_5 were reported in cattle and swine throughout Italy. In November 1985, serotype C FMD virus was diagnosed. A total of 68 outbreaks were recorded throughout the country by April 1986, mostly in swine. In June 1986, serotype A FMD virus was again diagnosed in Italy and an outbreak of serotype O FMD virus was reported. Type O had last been reported in Italy at Palermo in 1979. Since June 1986, additional outbreaks of type A FMD have been recorded in the northwest, northeast, and central regions of the country. More swine have been affected than cattle.

As a result of the Italian situation, the European Community (EC) Veterinary Committee held a meeting in Brussels, Belgium, on Sept. 2, 1986. Highlights of this meeting were reported as follows: The EC imposed a 3-month ban on Italian meat from all Italian counties where cases of FMD have been confirmed. If FMD is not controlled within this period, the ban will continue. The ban will be imposed on other counties where new FMD outbreaks are confirmed. Areas in Italy where FMD has not been confirmed can export only deboned meat, canned meat, and cured meat (that is, Parma ham) to the EC. Exports of livestock from Italy to the other member States of the EC are banned indefinitely. In addition, Yugoslavia has banned the importation of all Italian livestock and meat.

No outbreaks of FMD have been reported in Spain since the June 16, 1986, outbreak of subtype A_5 in that country. All markets have been reopened and it appears that the situation is under control.

Malaysia reported FMD serotype $Asia_1$ in wild fauna following outbreaks in domestic livestock that began in June 1985. The Pirbright World Reference Laboratory for FMD has confirmed serotype O in samples received from Hong Kong, Nepal, Saudi Arabia, Kuwait, and South and North Yemen.

Tanzania reported occurrence of FMD serotypes SAT2 and O. Cameroon reported serotype A. Samples from Burundi contained serotype O.

In South America, FMD serotypes A and C were reported from Ecuador, serotype C from Uruguay, and serotype O from Paraguay.

Hog cholera (HC) has not been reported in Great Britain since it was last confirmed on June 25, 1986. The virus was identified on a total of 10 premises and animals on an additional six premises were serologically positive. All the swine were depopulated. It now appears that bacon that had originated on the continent and that was subsequently fed as uncooked garbage was responsible for the primary outbreaks. The source of the bacon could have been any one of three countries: Holland, Belgium, or West Germany. British veterinary officials are confident that they have established the source of the problem and that the disease has been eradicated.

There has been an upsurge of hog cholera in Belgium, with 17 occurrences since September, bringing the total to 67 since the beginning of 1986. Austria, France, East and West Germany, and Italy continue to report HC. The disease was also reported in Malaysia (Sabah) and Taiwan.

In Africa, Togo has reported three outbreaks of **Rinderpest.** In one, 6 animals died and 10 were slaughtered. Control measures were reportedly taken in all cases. Rinderpest was also reported in Egypt.

The United States is considering Belgium's request for recognition as being free of **African swine fever (ASF).** The last report of ASF in Belgium was May 2, 1985. In the Netherlands, no outbreaks of ASF have been reported since April 1, 1986 (see 14-3). Italy (Sardinia) and Portugal continue to report ASF. (Dr. James T. Cavanaugh, 301-436-8285)

New Cattle Disease in Japan

Enzootic congenital abnormalities of unknown origin have been reported in cattle in southern Japan since November 1985. Abnormal newborn calves were reported in seven

prefectures of Kyushu Island, mainly in the southern part.

Reported Cases of Enzootic Congenital Abnormalities of Cattle by Prefecture and Month, 1986

Prefectures	Nov	Dec	Jan	Feb	Mar	Apr	May	Total
Fukuoka			1	4	8	3	1	17
Saga			1	2	1		1	5
Nagasaki			1	5	11	10		27
Kumamoto	2	7	24	32	44	6		115
Oita			27	56	67	21	8	179
Miyazaki			62	101	59	3	1	226
Kagoshima	54	274	650	517	149	34	26	1,704
TOTAL	56	281	766	717	339	77	37	2,273

At first glance, affected newborn calves may appear normal. However, some lack the ability to suckle or they suckle slowly and are weak. Some show nervous signs. Underdeveloped cerebrums and cerebellums have been observed at necropsy. However, no arthrogryposis, typical of Akabane disease, has been observed. Although the new disease has primarily affected beef cattle, it also has been seen in some dairy breeds. Characteristic congenital abnormalities have been recognized in cattle herds previously vaccinated against Akabane disease. No relationship has been extablished between the new disease and the reproductive history of dams.

The British National Institute of Animal Health (NIAH), Ministry of Agriculture, Forestry and Fisheries (MAFF), has been cooperating with Japanese prefectural government institutions, especially Livestock Hygiene Service Centers, in carrying out research to identify a cause of the abnormalities. Bacterial and viral examinations have not identified an etiological agent in nervous tissues and other organs of affected calves. Perivascular cuffing was observed microscopically in the brain, suggesting that the disease may be caused by an infectious agent. Skeletal muscle atrophy has not been observed, in contrast to the muscular damage frequently associated with Akabane disease.

Serological tests were conducted on fetal serum samples collected from calves showing such abnormalities using about 30 different strains of arboviruses; however, no reactions were observed. Antibodies in precolostral serum samples of affected calves were demonstrated against viruses which had been isolated from a blood sample collected from sentinel cattle kept at the Kyushu Branch Research Station of NIAH. These viruses were also isolated from culicoides there in the previous year. The viral isolates were kept at the research station in isolation from cases of enzootic congenital abnormalities. Research is in progress to characterize

the virus. Preliminary results have identified the isolates with a member of the orbivirus group of the Reoviridae family.

The NIAH has cooperated in nationwide surveillance to assess the disease situation. The surveillance revealed 3,243 cattle including 2,159 cases in Kyushu with congenital CNS symptoms or weakness, without other physical abnormalities, during the period between November 1985 and March 1986. Other diseases were found, including Akabane in Japan and bovine viral diarrea (BVD) in 47 calves in Hokkaido. (Excerpted from O.I.E. Asian Information No. 58, Asian Bureau of the Office International des Epizooties, Tokyo, Japan, by Dr. James T. Cavanaugh, 301-436-8682)

Screwworm Program Update

The Mexico-United States screwworm eradication program was plagued by several isolated outbeaks north of the sterile fly barrier at the Isthmus of Tehauntepec in Mexico during 1985. (See June 1985 issue, 13-2 for previous update.) Most of these were associated with animal movement from an infested area in southeast Mexico. The largest outbreak occurred in the States of Tamaulipas and San Luis Potosi where 139 confirmed screwworm cases occurred between June 25 and Aug. 8, 1985. The origin of this outbreak was not established; however, animal movements or intentional transport of fertile screwworm insects was suspected. The last two cases to be reported north of the barrier were collected from animals that had been transported from southeast Mexico to the state of San Luis Potosi in November 1985.

Inspection and quarantine stations located on the only highways crossing the Isthmus of Tehuantepec began operations prior to January 1985. All animals being transported across the Isthmus by these routes or by either of the two railroads are inspected, sprayed, or dipped in an effective larvicide, and larvicide is applied locally to any wounds. Infested animals are quarantined for 3 days while wounds are treated and observed. Quarantine enforcement is augmented by the presence of Mexican military personnel at each station.

An extensive information campaign was initiated in November 1985 for transporters and livestock owners at the points of origin of animal shipments. They were encouraged to inspect animals at points of origin and avoid transporting animals infested with screwworms. The result was a reduction in the number of infested animals presented at inspection and quarantine stations. From October 1985 to September 1986, 326,514 animals were inspected and 163 quarantined due to screwworm infestation. No screwworms have been found north of the sterile fly barrier since November 1985.

In August 1985, the Mexico-United States Commission for the Eradication of Screwworms approved the development of an amendment to the 1972 agreement which formed the Commission. The amendment authorized the Commission to extend the program into the Yucatan Peninsula, cooperate with international organizations, and negotiate and sign agreements with countries of Central America and Panama for the eradication of screwworms. The amendment was ratified by Mexico and the United States on April 1, 1986. An eradiction program in the Yucatan Peninsula began on May 19, 1986. By September, the numbers of reported cases from that area had been reduced. Most of the cases were near the border of Belize and Guatemala.

There have been discussions with the Governments of Guatemala and Belize about a screwworm eradication program. Agreements between the Mexico-United States Commission and those countries have been drafted and final negotiations are in progress.

The construction of a new plant for the production of sterile male screwworm flies in Panama is being planned. The new plant will have a capacity of at least 200 million sterile screwworm flies per week. An agreement with Panama is being drafted. Contracts with a construction management company and an architectural and engineering company will be awarded by the end of 1986. (Dr. J. E. Novy, Mexico-U.S. Commission for the Eradication of Screwworms, Apartado Postal M-2890, 06000 Mexico D.F., Mexico, 905-250-5992)

Research at Plum Island

(Note: The following report is the first of a series on foreign animal disease research at the Plum Island Animal Disease Center (PIADC), Agricultural Research Service, U.S. Department of Agriculture, Greenport, N.Y.)

Survival of several swine viruses in Parma ham was studied to determine the safety of importing this specialty product into the United States.

The name "Prosciutto di Parma" (Parma ham) is reserved exclusively for ham having characteristic qualities related to the geographical area of its production and the various phases of preparation from salting to the end of the curing period. Preparation and curing are carried out in a designated area within the Province of Parma, which is located in the Po Valley in northern Italy. The ham is obtained from heavy adult castrated male swine reared under controlled conditions. The curing period must not be less than 12 months. The United States Government banned the importation of these hams because of outbreaks of African swine fever (ASF) and swine vesicular disease (SVD) in Italy between 1973 and 1978. Cooperative studies were undertaken at PIADC, the Instituto Zooprofillattico Sperimentale, Brescia, Italy, and the Instituto Zooprofillattico Sperimentale, Sassari, Italy, to determine if the viruses of FMD, SVD, ASF, and

hog cholera (HC) were inactivated by the processing and curing procedures used in preparing Parma hams. Hams were prepared from pigs infected with each of these diseases when the carcass contained the greatest amount of virus. Thus, these experiments are examples of a "worst case situation," and in general hams would contain more virus than would probably occur under field conditions. SVD virus was not recovered after 360 days, FMD virus after 170 days, ASF virus after 399 days, and HC virus after 313 days. The processing and maturing of the ham inactivates these viruses and such hams, if processed through a 400-day period, they would be unlikely to be a source of disease transmission. (Dr. P. D. McKercher, PIADC, Greenport, N.Y., 516-323-2500)

Focus on...
Venezuelan Equine Encephalomyelitis

Venezuelan equine encephalomyelitis (VEE) is an acute, infectious disease of equines and humans caused by an arthropod-borne virus classified in the alphavirus group (group A arbovirus) of the Togavirus family. The disease is transmitted by mosquitoes and possibly other hematophagous insects. The disease was first reported in South America, where it is known as "peste loca." Horses, mules, and donkeys are equally susceptible to VEE virus.

History

VEE virus was first isolated in 1936 by guinea pig inoculation of a brain suspension from an encephalitic horse in Venezuela. The virus was shown to be different from Western (WEE) and Eastern (EEE) equine encephalomyelitis viruses in cross-protection studies in guinea pigs. Review of the literature suggested that VEE probably has occurred in Venezuela, Colombia, and Peru since at least the early 1900's. Epizootics of VEE occurred yearly from 1936-73, with major peaks of activity at 6- to 10-year intervals. Tens of thousands of equines were clinically affected and thousands of deaths occurred during peak periods. The first VEE-related human illness was reported during a major outbreak in Trinidad in 1944. Mansonia titillans mosquitoes were incriminated as the virus vector. Outbreaks in Venezuela during the late 1950's and 1960's and laboratory infections among researchers confirmed that VEE virus was pathogenic for humans. Infection in humans produces a "flu-like" illness that has been described as a "most unpleasant illness, memorable for years." Encephalitis and deaths were most commonly reported in young children and adolescents.

The most recent VEE activity began in Venezuela and Colombia in 1969 as a devastating epizootic that spread to Ecuador and Peru. In an unprecedented occurrence, VEE virus was transported in an unknown manner to an area on the Pacific Coast of the border between Guatemala and El

Salvador in late June 1969. An epizootic wave of VEE virus activity moved relentlessly through the equine population of all Central American countries (except Panama) and Mexico, eventually reaching the United States (Texas) in late June 1971. Approximately 142 equines in the United States were confirmed to have died from VEE during this activity in 1971. The last documented cases of VEE in North America occurred in Mexico in 1972. In South America, the last cases of VEE in equines occurred in the Goajira region of Venezuela in 1973. The 1969-1972 epizootic of VEE was modified and ultimately interrupted by the use of an attenuated VEE vaccine and aerial spraying of ultra-low volume (ULV) malathion over millions of acres of land in Texas and Mexico. Epizootic VEE activity has not been reported worldwide since 1973.

Geographic Distribution

Epizootic VEE has occurred naturally in Venezuela, Colombia, Trinidad, Peru, Ecuador, Costa Rica, Nicaragua, Honduras, El Salvador, Guatemala, Belize (formerly British Honduras), Mexico and the United States (Texas). Some epizootics in Argentina and Venezuela have been ascribed to equine vaccination with an incompletely inactivated formalin-treated VEE virus vaccine. Epizootic VEE has been associated with equines in areas of Latin America ecologically classified as tropical dry or tropical thorn forests. These areas have prominent dry seasons and are the traditional cattle-raising and rice-growing areas of the tropics. Here VEE epizootics may continue with the presence of mosquito vectors as long as susceptible equines are available. Human infections typically follow equine disease by approximately 2 weeks. Humans are tangentially infected from equines; epizootics and epidemics cease when susceptible equines are no longer available.

During the 1960's, VEE virus activity was reported in areas of tropical and subtropical America where VEE epizootics in equines had never been reported. Occasional human illnesses and fatalities confirmed the pathogenic nature of the VEE isolates; however, equine disease did not occur.

Virus Subtypes and Variants

In a classical study, Young and Johnson (Amer. J. Epidemiol. 89: 286, 1969) applied a short incubation hemagglutintion inhibition (HI) test by using immune sera produced by the inoculation of the spiny rat, Proechimys semispinosus, to test all available isolates of VEE virus. The antigenic relationships observed were stable upon passage and were not related to the donor host from which the virus was isolated. They showed that place of origin and, to a lesser extent, time were important determinants of antigenic variation, that isolates could be differentiated geographically and that certain isolates were associated with epizootics, while others were not. These researchers separated the isolates into

four subtypes (I-IV); and with subtype I, they identified five variants (A-E). Later studies identified subtype I variant F, two variants within subtype III (A and B), and two additional subtypes (V and VI). These studies confirmed the Young and Johnson distribution, except for subtypes I-A and I-B, which were shown to be identical and are now referred to as I-AB. Young and Johnson recognized that all VEE isolates associated with equine disease were antigenically grouped as subtype I, variants, A, B, and C (epizootic variants). These variants have only been isolated during equine epizootics; isolations ceased when there was no more epizootic activity. It was presumed in 1969 that an equine epizootic with the other subtypes/variants awaited only the presence of competent vectors and susceptible equines.

Prior to 1969, variants I-AB and I-C were isolated only during equine epizootics in northern South America (Venezuela, Trinidad, Colombia, Ecuador, Peru, and Argentina). In contrast, the other subtypes and variants were demonstrated continuously over time in areas where few equines or humans ventured, tropical wet forests and shaded fresh-water swamps. Such areas included the Florida Everglades (subtype II); discontinuous foci from Veracruz, Mexico, to Almirante, Panama (variant I-E); an area from the Panama Canal Zone to Colombia and Venezuela (variant I-D); and Trinidad, Brazil, Suriname, and Guyana (subtypes III-A, III-B, and IV). While occasional outbreaks of VEE occurred in humans, equine VEE was not reported. In later studies, variant I-F was demonstrated in Brazil; subtype III-B was found in an unusual association with swallow bugs and birds in ecologically arid areas of Colorado and Utah; subtype V was isolated in French Guiana; and subtype VI was isolated in Argentina.

With the outbreak of VEE in Central America in 1969, it was initially presumed that the long-expected epizootic expression of variant I-E had occurred. The revelation that the epizootic variant was I-AB, identical to the epizootic virus concurrently active in Ecuador at that time, forced a reevaluation of VEE viruses. It is now accepted that the variants I-D, I-E, and I-F, and subtypes II, III, IV, V, and VI are sylvatic viruses found in enzootic foci. **The sylvatic VEE viruses do not cause VEE in equines, but are pathogenic for humans.** There are no data to suggest that epizootic (equine virulent) VEE variants arise from sylvatic VEE viruses in enzootic foci. While epizootic VEE and the epizootic VEE variants have not occurred since 1973, the sylvatic VEE viruses in enzootic foci currently persist in many areas of subtropical America.

Transmission

The primary means of transmission of both epizootic and sylvatic VEE viruses is by mosquitoes. Vector mosquitoes

are found in virtually every aquatic habitat in tropical America. The sylvatic viruses circulate among rodents and birds by the feeding activity of mosquitoes primarily in the genus Culex subgenus Melanoconion. Humans and equinea seldom venture into the enzootic focus of activity.

Infection of humans with either sylvatic or epizootic VEE viruses can produce severe clinical disease, encephalitis, and death. Infection of equines with sylvatic VEE virus causes low level viremias but no clinical disease; infection does, however, confer cross protective immunity in equines to subsequent infection with epizootic VEE virus.

Due to the high concentrations of viremias in infected equines, epizootic VEE viruses can be transmitted by many species of hematophagous insects. At least 34 species of 8 genera of mosquitoes have been implicated as biologic vectors of epizootic VEE viruses. The most important epizootic VEE virus vectors are Mansonia titillans, M. indubitans, Psorophora confinnis, Ps. discolor, Aedes taeniorhynchus, A. sollicitans, A. aegypti, A. thelcter, A. scapularis, and Deinocerites pseudes. In addition, Simulium spp. and Culicoides spp. have been suggested as biologic or mechanical vectors. Epizootic VEE virus can be moved vast distances in a short period of time.

Typically for arboviruses, including VEE virus, transmission occurs with the feeding of infected female mosquitoes on susceptible hosts. The virus must replicate during an extrinsic incubation period in the biological vectors before virus can be transmitted via the salivary glands to the host.

While aerosol transmission to humans commonly occurs in laboratory-acquired infections, aerosol or direct contact transmission in naturally infected equines is negligible. Natural human infection with epizootic VEE virus occurs tangentially from the feeding of vector mosquitoes on infected equines and subsequently on humans.

Pathogenesis

Within 12 to 24 hours after infection, a febrile response and viremia occur in equines. Fever may reach 104°F (40°C) and viremia may exceed $10^{5.5}$ $SMICLD_{50}$ (suckling mouse intracerebral median lethal doses)/ml of blood.

Viral titers may occasionally exceed $10^{8.8}$ $SMICLD_{50}$/ml. At approximately 5 days after the onset of infection, viremia ends, neutralizing antibodies can be demonstrated, and initial signs of central nervous system (CNS) disease appear. Clinical signs may vary from a mild, febrile response to severe CNS disease with stimulation, hyperexcitability, hyperesthesia, profound depression, and death. Signs may also include leukopenia with profound lymphopenia, psychic depression with

drooping ears, and a somnolent appearance, trismus, inability to swallow water, frothing at the nostrils and mouth, anorexia, walking in circles and into obstacles, and pressing of the head against obstacles. Death may occur within 5 to 6 days or as late as 9 to 14 days after the onset of infection. Animals with mild infections may recover and have no neurologic sequelae.

Lesions

In the equine, VEE causes lymphoreticular destruction and encephalitis. Visible lesions are the result of self-inflicted trauma. Lesions may include contusions and abrasions on the skin and hemorrhages of the surface of the brain and meninges. Microscopic changes include encephalomyelitis and hemorrhages of all types in the brain and meninges.

Diagnosis

A tentative diagnosis of viral encephalomyelitis in equines may be based upon the occurrence of acute neurologic disease during the season in which biting fly activity occurs and in the presence of large numbers of mosquitoes and hematophagous insects. Typically, VEE virus will cause clinical disease in many equines at the same time rather than cause individual cases. Confirmtory diagnosis during an epizootic of equine encephalomyelitis must be based upon isolation and identification of virus or upon seroconversion demonstrated with the complement fixation, hemagglutination inhibition, or neutralization test. The differential diagnosis of VEE includes eastern and western equine encephalomyelitis and other less frequently reported arbovirus-caused encephalitides, rabies, African horsesickness, equine infectious anemia, toxic encephalitis, mineral poisoning, botulism, shock, hepatoencephalopathy, protozoal and verminous encephalitis, and leukoencephalomalacia.

Due to the occurrence of sylvatic VEE virus in many parts of tropical America, a diagnosis of VEE in individual equines based upon serologic studies alone must be made with extreme caution. Sylvatic VEE virus infection in equines will produce antibodies against epizootic VEE viruses that can be differentiated only by the viral neutralization test.

Virus isolation is most easily completed by intracranially inoculating suckling mice or inoculating cell cultures with blood collected from febrile, clinically normal equines located near encephalitic equines. VEE virus is then identified by the HI or neutralization test.

Control

During the most recent epizootic in 1969-72 in North America, control was effected by a combination of vector control by the aerial application of insecticide and vaccination of equines.

The vaccine used had been earlier developed for use in humans. The use of a human vaccine in equines was unprecedented in the annals of virology. This live, attentuated vaccine, strain TC-83, proved to be safe and effective for equines when properly applied. Millions of equines were safely vaccinated during and after the outbreak. Immunity to epizootic VEE infection occurs within 5 days after vaccination and probably persists for life.

A formalin-inactivated VEE vaccine produced from virus replicated in embryonated chicken eggs was used for decades in Latin America. Experience has shown that VEE virus is exceptionally difficult to inactivate. Numerous outbreaks of VEE have been traced to the use of such formalin-treated preparations. Formalin-inactivated VEE vaccines made with virulent or nonattenuated viruses should never be used in equines or humans.

A trivalent VEE, EEE, WEE formalin-inactivated vaccine is commercially available in the United States. The VEE virus antigen used is strain TC-83. The vaccine is safe and effective.

Summary

Clinical VEE in equines is caused only by certain variants of VEE virus. Epizootic VEE virus has not been isolated and clinical VEE has not been confirmed in equines since 1973. Sylvatic variants and subtypes of VEE virus are commonly isolated in tropical and subtropical America. Sylvatic strains of VEE virus may cause clinical encephalitis in humans, but are nonpathogenic for equines. (Dr. Thomas E. Walton, USDA, ARS, Arthropod-borne Animal Diseases Research Laboratory, P.O. Box 3965, University Station, Laramie, WY 82071)

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